LEAD-POISONING WITH SPECIAL REFERENCE TO THE SPINAL CORD AND PERIPHERAL NERVE LESIONS.

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In recording this case, I do so with the object of adding to the testimony already collected in support of the general rather than the local affection of the nervous system by lead-poisoning.

Many cases have now been recorded in which microscopical investigation has revealed that the central nervous system is involved, as well as the peripheral nerves.

The question as to whether the former is secondary to the latter, is still unanswered.

Those favoring the central disease as the primary cause of the peripheral lesions, claim that disease of the anterior gray matter of the cord exists, although not apparent to our present modes of examination.

It would, however, seem reasonable to suppose, as Pal has suggested, in his monograph on multiple neuritis, that toxic agents such as lead, alcohol, arsenic, etc., act simultaneously on the whole nervous system, especially involving, perhaps, the peripheral nerves, but also involving the central nervous system.

In such cases, however, where recovery does not take place early in the disease, that is, in those pursuing a chronic course, or when the toxic agent is still in force, it would appear that the central nervous system becomes affected, and its disease manifests itself in well-recognized organic and systemic diseases.

This explanation would seem to accord with the clinical history of many of the cases of multiple neuritis, especially of alcoholic origin, when so-called pseudo-tabes and psychical derangement are prominent symptoms.

A basis for this permanent condition is not infrequently found outside of the peripheral nerves in disease of the meninges and substance of the brain and cord.

Present investigation would indeed seem to show that disease strictly limited to certain systemic tracts, so called, is rarer than formerly supposed, and that a diffuse affection of various columns and of various areas is common. This would seem also the more reasonable, as clinically, even in such a frequently observed and familiar organic disease as tabes, many symptoms are either absent, which are considered diagnostic of the disease, or again, others are present which are directly opposed to the situation of the lesion in the posterior columns. There has never been any

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satisfactory explanation, to my mind, of the selection of certain tracts with limitation to their sole environment by disease of infectious or toxic nature. The common selection of the upper extremities, as the seat of disease in lead-poisoning, and especially of the extensor group of muscles of the forearm, and those of the hand, may possibly be explained on the ground that those muscles are especially exposed to over-use.

I have observed that those cases of lead-poisoning more strictly limited to the muscles of the hands, resembling in its distribution that of progressive muscular atrophy, are usually difficult of cure, if indeed much improvement takes place at all. I have considered that in these cases, a spiral lesion involving the anterior horns was probably present, although hitherto unable to support the theory by autopsy.

The present case, which I now present, was of that nature, and the findings would seem to support my clinical deduction. In lead-poisoning, however, we do not always find the paralysis limited to the upper extremity. The lower extremities may be affected, or the paralysis may be hemiplegic in its distribution. Buzzard, in his work on *Disease of the Nervous System*, Lect. XXV., on "Some Points in the Diagnosis of Lead Palsy," relates:

> A woman, aged twenty-five years, had paralysis of the forearm and hand muscles of the right side, and of those of the leg of the left side, with foot-drop. There was loss of faradic response, and a reaction of degeneration to galvanism; the left forearm also showed less response to faradism than normal, although no paralysis had been demonstrated. The case ended in recovery.
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> In a second case, also a woman, with paralysis of all the extremities, there was loss of patellar reflex, and loss of reaction to both faradism and galvanism.
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> A third case, a sign-painter exposed to lead, and exhibiting the blue line (although not a very characteristic one), presented at first a weakness of the left forearm and both legs, and later, of the right forearm, although the right side showed no perceptible alteration. There was also wasting of the interossei of the left hand. There was loss of excitability to faradism in the wasted muscles, and in the supinators. (From this latter fact, Dr. Buzzard mainly makes the diagnosis of progressive muscular atrophy.) The autopsy revealed "extensive atrophy of the large ganglionic cells in the anterior horns (more marked on one side than the other), in the cervical and dorso-lumbar regions, together with what was apparently a commencing overgrowth of connective tissue in the lateral columns.

I have related the last case quoted from Dr. Buzzard rather fully, as in many respects it resembles my own, and as supporting the theory of the affection of the central nervous system in lead-poisoning. The etiology in this case I would ascribe to lead, and would therefore differ from the author as to the diagnosis.

I will briefly give the history of the case on which I have based this paper:

A. E., painter, first seen June, 1890. Family history negative. *Previous history:* He has worked at his trade since the age of thirteen;

when twenty-one he had an attack of painter's colic, with some weakness of the arms. Two years later he had a second attack, since which time there have been several mild attacks. For the past two years he has become progressively weaker in his hands, which have become wasted, so that he is unable to hold his brush. Three months ago, he had an epileptic seizure, in which he lost consciousness, and was taken to Bellevue Hospital. He remained there for three months, and improved but slightly.

Present condition: Patient anæmic, and sallow. Complains of weakness in both forearms and hands, especially of the latter, which present the appearance seen in progressive muscular atrophy. Heart hypertrophied; radial arteries, as also the temporals, show high tension. Eyesight poor; patient is unable to read well unless with a bright light. Ophthalmoscopic examination by Dr. C. S. Bull showed the following: R. E., 20/xxx; L. E., 20/c; irregular hemianopsia, the nasal half of the field being intact. R. E.: Exudative neuro retinitis, with intense exudation over the disc and neighboring portions of the retina. L. E.: Atrophy of the optic nerve and retina, with occlusion of some of the vessels. Suspected degenerative nephritis. Examination showed marked loss of power in both forearms and hands, more marked on the right side. Great atrophy of the interossei, and of the muscles of the ball of the thumbs. Grasp feeble; no tremor; able to extend the hand with great difficulty. Sensation lowered to heat and cold, and to touch and pain, and, to a slight degree, to the faradic current. Electrical examination: The extensors of the forearm show excitability to the faradic current, but much reduced. Supinators unaffected. Muscles of the hands, the interossei and opponens pollicis show no response to faradism. There was a loss of excitability to galvanism, but no reaction of degeneration. There was no affection of the lower extremities, nor was there rectal or vesical paralysis. Reflexes were normal; no pain on pressure along the course of the nerve or in the muscles. Urine: sp. gr. 1008; albumin.

He was put on potass. iodid. and iron, with galvanism, three times a week.

There was no change in the condition of the muscles during the following ten months, either in power, or in response to the electrical currents; but as the Bright's disease progressed, he entered Bellevue Hospital again, where he died, March, 1891.

Autopsy.—Heart hypertrophied; lungs normal; kidneys the seat of interstitial nephritis; brain normal, except that the arteries were the seat of atheromatous changes. The cord, including the cervical and upper dorsal portions, and the musculo-spiral nerve were hardened in Müller's fluid, and examined microscopically by Dr. W. Coleman, of the Loomis Laboratory. The peripheral nerves were markedly degenerated. The cord at the upper dorsal region showed some atrophy of the anterior horns, especially of one side, and also of the antero-lateral tracts of that side. In certain levels, there was evident sclerosis of the column of Goll, and also in Lissauer's tract there was thickening of the meninges and of the bloodvessels. In the substance of the left anterior cornua there was an apparent breaking down, leading to loss of substance. This was probably increased artificially in the examination. The anterior nerve roots also showed degeneration. While these changes are not extensive, still the evidence is clear, of a slow, progressive, degenerative process.

We have, then, changes indicative of sclerosis, disseminated in character. The whole body was evidently the seat of like changes, as shown in the arteries and kidneys.

This case is somewhat similar to the one recorded by Pal, both in its history and microscopical findings—see his monograph on *Multiple Neuritis.*

CASE IV. The patient, aged twenty-one years, showed in his first attack, colic and convulsion. A transient paralysis of the right abducens and facial, and later, of the left side. Papillitis on both sides. Hemorrhage in the right eye, the vessels, especially the veins, being strangulated. Patient could distinguish light from darkness; upper extremities ataxic and weak; lower extremities, atrophic; patellar reflex absent. Sensibility reduced.

Autopsy.—Showed partial degeneration in the optic, oculo-motor, abducens, facial, right radial, and left peroneal. The cord showed degeneration of the white columns of a disseminated character, as also of some of the cells in the anterior horn, and the posterior nerve roots.

In conclusion, if I understand the author correctly, he finds in his case a disseminated sclerosis, all the tracts of the cord being affected in different areas, and from this, and other cases, he holds that multiple neuritis affects the nerves in the tract of the cord in the same manner as it does the peripheral nerves.

From the literature of the subject (for reference to which I would refer again to Dr. Pal's monograph), and from the case I have referred to, it is evident that the various toxic agents, as arsenic, lead, alcohol, etc., cause multiple neuritis, involving at times the upper and lower extremities, although with each individual agent certain regions are more often selected; and also that with the peripheral lesion there is not infrequently associated organic lesions of the central nervous system.

The frequent association of cranial nerve lesions in lead-poisoning, as well as from other toxic agents in which we find changes in the nerves, or their nuclei, should have prepared us to expect like lesions in the gray matter of the cord, and as these paralyses sometimes occur with disease of the nerve alone without the involvement of the nuclei, so, to carry out the analogy, it would seem impossible for it to be otherwise in the relation of the peripheral nerves to the spinal cord.

An interesting question is suggested by Pal in reference to the trophic dependence of the motor spinal nerves to the cells of the anterior horns. He finds that in some cases of atrophy of the cells, no degeneration of the nerve fibre followed. The subject would lead me into a discussion of poliomyelitis, which has already occupied considerable of the time of the session, and therefore I will only say that this sustains again the fact of the simultaneous affection of the peripheral and central nerves as well as their individual, and, at times, exclusive disease.